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Issue Date: 29 August 2006

CASE NO.: 2004-BLA-0072

In the Matter of

E. M., Survivor of
M. D., SR., Deceased Miner,
Claimant,

v.

ENERGY WEST MINING COMPANY
(now d/b/a UTAH POWER & LIGHT, MINE DIVISION)
Employer,

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS,
Party-in-Interest.

Appearances:

Martin J. Linnet, Esquire,
For the Claimant

William S. Mattingly, Esquire,
For the Employer

Before: WILLIAM DORSEY
Administrative Law Judge

DECISION AND ORDER ON REMAND AWARDING BENEFITS

This matter is before me on a remand from the Benefits Review Board ("Board") dated February 27, 2003 from a Decision and Order Awarding Benefits issued on November 16, 2001, by Administrative Law Judge Paul H. Teitler. Judge Teitler awarded the widow benefits because he found that the miner had pneumoconiosis through both x-ray and medical opinion evidence; that his pneumoconiosis arose out of his coal mine employment; that he was totally disabled based on the pulmonary function, blood gas studies, and medical opinions; and that his disability was due to pneumoconiosis. Judge Teitler also found that the miner had established a material change in condition.

Also critical to this remand decision is the order of December 6, 2000 that Judge Teitler issued after the hearing but before his decision that arranged to obtain an "impartial" reading of an August 18, 2000 x-ray.

Employer appealed Judge Teitler's Decision to the Board, contending that he erred in: (1) obtaining and admitting Dr. Barrett's post-hearing x-ray report; (2) applying the duplicate claim standard; (3) finding the existence of pneumoconiosis pursuant to §§ 718.202(a)(1) and (4); and finding that the miner was totally disabled due to pneumoconiosis. In its unpublished decision of February 27, 2003, the Board affirmed in part, vacated in part, and remanded the case for further consideration. The Board held that it was error not to provide Dr. Barrett's x-ray reading to the parties for comment or rebuttal before the Decision and Order was issued. The Board also held that Judge Teitler erred in applying the Tenth Circuit's duplicate claim standard as set forth in *Wyoming Fuel Co. v. Director, OWCP [Brandolino]*, 90 F.3d 1502, 20 BLR 2-302 (10th Cir. 1996). It remanded the case so that the evidence obtained after the denial of an earlier claim could be compared to the evidence used to adjudicate the earlier claim; the comparison would show whether the miner had established a material worsening of the two elements previously adjudicated against him: the existence of pneumoconiosis and disability causation. Only if there were a material change with respect to both those elements could the merits of the new claim be addressed.

The parties agreed that a further trial was unnecessary. The record consists of Director's Exhibits (DX) 1-54, Claimant's Exhibits (CX) 1-13, and Employer's Exhibits (EX) 1-20, all of which are now admitted into evidence.

Duplicate Claim Standard in the Tenth Circuit

The miner's last coal mine work was in Utah, so the interpretation of the Act and the regulations by the United States Court of Appeals for the Tenth Circuit controls. *Shupe v. Director, OWCP*, 12 BLR 1-200 (1989). The Tenth Circuit held in *Brandolino*, 90 F.3d 1502, that to show a material change in conditions under § 725.309, a claimant "must prove for each element that actually was decided adversely to [him] in the prior decision that there has been a material change in that condition since the prior claim was denied." The court stated:

> In order to meet the claimant's threshold burden of proving a material change in a particular element, the claimant need not go as far as proving that he or she now satisfies the element. Instead, under the plain language of the statute and regulations, and consistent with res judicata, the claimant need only show that this element has worsened materially since the time of the prior denial. . . . However, a new interpretation of an old x-ray that was taken before the prior denial or a further blood gas result identical to the results considered in the prior denial does not demonstrate that a miner's condition has materially changed.

Therefore, the Claimant must offer proof of a material change in the evidence about whether the miner (1) had pneumoconiosis and (2) was totally disabled by pneumoconiosis.

Existence of Pneumoconiosis

The Regulations define pneumoconiosis broadly as "a chronic disease of the lung and its sequelae, including respiratory and pulmonary impairments arising out of coal mine employment." 20 C.F.R. § 718.201. It is progressive and irreversible. *Woodward v. Director, OWCP*, 991 F.2d 314, 320 (6th Cir. 1993). The compensation program recognizes both medical, or "clinical," pneumoconiosis and statutory, or "legal," pneumoconiosis. *Id.*; *Anderson v. Director, OWCP,* __ F.3d __, 2006 WL 2053841 at *1 & n.2 (10th Cir. 2006). Clinical pneumoconiosis comprises:

> Those diseases recognized by the medical community as pneumoconioses, i.e., the conditions characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. This definition includes, but is not limited to, coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis, or silico-tuberculosis, arising out of coal mine employment.
> 20 C.F.R. § 718.201(a)(1).

Legal pneumoconiosis, on the other hand, includes "any chronic lung disease or impairment and its sequelae" if that disease or impairment arises from coal-mine employment. 20 C.F.R. § 718.201(a)(2). A claimant's condition "arises out of coal mine employment" if it is a "chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment." *Id.* Finally, the Regulations reiterate that pneumoconiosis is "a latent and progressive disease" that might only become detectable after a miner has ceased to be exposed to coal dust. *Id.*

Ordinarily more weight is given to the most recent evidence, *see Mullins Coal Co. of Virginia v. Director, OWCP*, 484 U.S. 135, 151-152 (1987); *Crace v. Kentland-Elkhorn Coal Corp.*, 109 F.3d 1163, 1167 (6th Cir. 1997), but the rule is not simplistically applied to require that later evidence be accepted over earlier evidence. *Woodward*, 991 F.2d at 319-320.

The Regulations provide four methods to prove that a miner suffers from pneumoconiosis: (1) chest X-rays; (2) autopsy or biopsy evidence; (3) the presumptions in §§ 718.304, 718.305, and 718.306; and (4) medical opinions finding that Claimant has pneumoconiosis. *See* 20 C.F.R. § 718.202(a)(1)-(4). As there is no autopsy or biopsy evidence and Claimant is not eligible for the presumptions,[1] only chest X-rays, CT scans, and medical opinions can establish the existence of pneumoconiosis in this claim.

X-ray Evidence Under § 718.202(a)(1)

[1] Claimant is ineligible for the § 718.304 presumption because he has not been diagnosed with complicated pneumoconiosis. Claimant cannot qualify for the § 718.305 presumption because he did not file this claim before January 1, 1982. Claimant is ineligible for the § 718.306 presumption because he did not die before March 1, 1978.

The chart below summarizes all the x-ray evidence in the record, including those x-rays taken in conjunction with the prior denial, those submitted with the duplicate claim, and those submitted in response to Dr. Barrett's x-ray report.

Chest X-rays

Exhibit No.	**X-ray Date**	**Physician/ Qualifications**	**Interpretation**
DX 31	8/21/73	Bauermeister/BCR	0/1; s
DX 31	6/7/79	Bauermeister/BCR	Lungs appear clear
DX 31	6/7/79	Sargent/B, BCR	Completely negative
DX 32	9/19/84	Bauermeister/BCR	Completely negative
DX 32	9/19/84	Sargent/B, BCR	Negative
DX 32	10/29/84	Bauermeister/BCR	Completely negative
DX 32	10/29/84	Sargent/B, BCR	Negative
DX 34	2/12/87	Sargent/B, BCR	Negative
DX 33	2/2/88	Sargent/B, BCR	Negative
DX 34	12/16/91	Mitchell	Unremarkable appearing chest with no significant change since previous exam of 2/12/87
DX 34	12/16/91	Sargent/B, BCR	Negative
DX 18	3/1/94	Baldwin/BCR	Clear lungs; no significant change since 11/2/84; negative chest
DX 35	9/6/94	Sheya/BCR	Negative; subpleural fat—mimics pleural thickening
DX 35	9/6/94	Lee/B, BCR	0/1; t/s; 2 lower zones
DX 18	10/5/94	Sheya/BCR	No change since March 1994; nonspecific irregular markings within lung bases that are likely related to age or smoking
DX 18	12/7/94	McClennan/BCR	Bilateral pleural thickening with obesity; no evidence of acute cardiopulmonary disease
DX 18	1/21/97	Baldwin/BCR	No acute abnormalities; questionable faint interstitial nodular densities in the lateral aspect of the right chest, which may have been present dating back to 3/1/94
EX 4	1/21/97	Branscomb	Negative
EX 5	1/21/97	Repsher/B	Progressive development of extremely severe, at first bibasilar and then generalized, interstitial fibrosis; UIP/IPF
DX 18	12/22/97	Baldwin/BCR	Minimal interstitial densities both mid and lower lung fields
EX 4	12/22/97	Branscomb	Negative
DX 18	1/7/98	Baldwin/BCR	Very minimal, subtle interstitial densities at both costophrenic angles, stable from 1994
EX 4	1/7/98	Branscomb	Negative

DX 18	12/14/98	Baldwin/BCR	Minimal vague densities lateral chest
EX 4	12/14/98	Branscomb	Negative
DX 24	9/2/99	Wheeler/B, BCR	Negative; 6 mm nodule or artifact in lateral periphery right upper lung or pleura at level of anterior rib-2
DX 24	9/2/99	Scott/B, BCR	Negative; few small calcified granulomata periphery of both lungs, probably healed histoplasmosis
DX 12	9/2/99	Navani/B, BCR	1/0; p/q; 5 zones; pleuro-parenchymal changes more likely secondary to asbestos than coal dust
DX 13	9/2/99	Sheya/BCR	2/2; t/r; 4 zones
EX 11	12/21/99	Wheeler/B, BCR	Negative; minimal linear and irregular increased lung markings in lower lungs and lateral periphery right mid lung with possible few tiny calcified granulomata compatible with healed histoplasmosis or possible usual interstitial pneumonitis (UIP)
EX 12	12/21/99	Scott/B, BCR	Negative; questioned moderate non-specific linear interstitial fibrosis and UIP
EX 4	12/28/99	Branscomb	Negative; probable congestive heart failure
ALJX 1	8/18/00	Barrett/B, BCR	1/2; q/r
CX 6	8/18/00	James/B	2/3; u/u; 5 zones
EX 17	8/18/00	Wheeler/B, BCR	Negative; moderate linear and irregular interstitial infiltrates or interstitial fibrosis in lower lungs and right mid lung and in lateral periphery left mid lung involving pleura with subtle lateral pleural fibrosis and thickening minor fissure compatible with inflammatory disease such as autoimmune disease or usual interstitial pneumonitis
EX 18	8/18/00	Scott/B, BCR	Negative; peripheral mid and lower lung interstitial infiltrates and/or fibrosis with or without edema; most likely UIP, possibly with superimposed CHF
EX 5?	8/18/00	Repsher/B	Unreadable
EX 4	1/18/01	Branscomb	Negative; pneumonia
EX 4	8/1/01	Branscomb	Negative
EX 4	8/10/01	Branscomb	Unreadable
EX 4	8/11/01	Branscomb	Unreadable but negative
EX 4	8/14/01	Branscomb	Negative
EX 4	8/15/01	Branscomb	Negative; congestive heart failure
EX 4	8/17/01	Branscomb	Negative
EX 4	8/20/01	Branscomb	Negative

EX 4	9/2/01	Branscomb	Unreadable
DX 45	9/3/01	Sharma	Stable opacities in both lungs
EX 4	9/3/01	Branscomb	Unreadable
EX 4	9/5/01	Branscomb	Unreadable
EX 5	9/5/01	Repsher/B	Progressive development of extremely severe, at first bibasilar and then generalized, interstitial fibrosis; UIP/IPF

Dr. Repsher believed from his review of the x-rays taken between January 21, 1997 and September 5, 2001 that the Claimant had developed a severe, at first bibasilar and then generalized, interstitial fibrosis. He later labeled this as usual interstitial pneumonitis/idiopathic pulmonary fibrosis (UIP/IPF). EX 5. He did not provide ILO reports of the x-rays he reviewed and did not specify exactly which x-rays he read other than the 1/21/97 and 9/5/01 films. The Board held that Judge Teitler erred in not including Dr. Repsher's classification of the August 18, 2000 x-ray as unreadable and directed that his interpretation be addressed on remand. While my review of the record did not reveal this reading, and, in fact, the Employer's summary of evidence attached to its brief on remand does not list this reading, I will consider it. Otherwise, I will only consider Dr. Repsher's interpretations of the 1/21/97 and 9/5/01 x-rays.

The newly submitted x-rays begin with the January 21, 1997 film. Prior to that, there were 16 readings of 11 separate x-rays taken between August 21, 1973 and December 7, 1994. None of those 16 readings were positive for pneumoconiosis. Fifteen of those interpretations were made by board-certified radiologists, two of whom are also B-readers. The x-ray evidence prior to the most recent duplicate claim was overwhelming negative for pneumoconiosis.

Since the most recent denial, there are 34 readings of 20 x-rays taken between January 21, 1997 and September 5, 2001. The January 21, 1997 x-ray was read by Dr. Baldwin, a board-certified radiologist, as showing no acute abnormalities, but he did question the presence of faint interstitial nodular densities. Dr. Branscomb, a lapsed B-reader, read this film as negative, and Dr. Repsher, a B-reader, found progressive development of interstitial fibrosis that he subsequently diagnosed as usual interstitial pneumonitis/idiopathic pulmonary fibrosis (UIP/IPF). Because the interpretations of Drs. Baldwin and Repsher are consonant, I place more weight on them than on Dr. Branscomb's simple negative reading. While they may not, in and of themselves, be considered readings of pneumoconiosis, they add to the persuasiveness of the medical opinion from Dr. James that is described below.

The December 22, 1997 x-ray was read by Dr. Baldwin as showing minimal interstitial densities in the mid and lower lung fields. Dr. Branscomb read this x-ray as negative. Once again, I place greater weight on Dr. Baldwin's opinion because of his status as a board-certified radiologist, but, by itself, I do not consider this x-ray positive for pneumoconiosis.

The January 7, 1998 x-ray was also read by Dr. Baldwin and Dr. Branscomb. Dr. Baldwin found minimal, subtle interstitial densities, while Dr. Branscomb felt the film was negative. Once more I place greater weight on Dr. Baldwin's opinion because of his greater training as a board-certified radiologist, but, by itself, I do not consider this x-ray positive for pneumoconiosis.

The December 14, 1998 x-ray was read by Dr. Baldwin as showing minimal vague densities. Dr. Branscomb read the x-ray as negative. Again, based on credentials, I defer to Dr. Baldwin's interpretation but do not consider it, by itself, positive for pneumoconiosis.

Four physicians read the September 2, 1999 x-ray. Dr. Wheeler regarded it as negative for pneumoconiosis but spotted a 6-mm nodule in the right upper lung. Dr. Scott thought the film negative for pneumoconiosis but saw a few small calcified granulomata in both lungs that he believed was probably healed histoplasmosis. Dr. Navani read the film as positive for pneumoconiosis with a reading of 1/0. Dr. Sheya opined that the film showed category 2/2 pneumoconiosis. All four physicians are board-certified radiologists, and Drs. Wheeler, Scott, and Navani are also B-readers, so all four potentially are entitled to great weight. I find Dr. Navani's reading, as supported by Dr. Sheya's, to be the most credible, and because both Dr. Wheeler and Dr. Scott found a nodule or granulomata, I find that their readings have some tendency to support Dr. Navani's interpretation. Accordingly, I find that this x-ray is positive for pneumoconiosis.

Drs. Wheeler and Scott read the December 21, 1999 x-ray. Both believed it was negative for pneumoconiosis but questioned whether the linear markings represented IP. I do not consider this x-ray, by itself, to be positive for pneumoconiosis, but it does lend weight to Dr. James's opinion that is discussed below.

The December 28, 1999 x-ray was read as negative by Dr. Branscomb, who thought that it probably showed congestive heart failure. It was not read by any other physician of record. Accordingly, I consider it negative.

The August 18, 2000 x-ray was one that Dr. Repsher, a B-reader, found unreadable. Yet four other B-readers, and three of whom are also board-certified radiologists, did read it, which leads me to reject Dr. Repsher's position about its quality. Dr. Barrett and Dr. James read the x-ray as positive for pneumoconiosis. Dr. Barrett found category 1 pneumoconiosis, while Dr. James categorized the film as stage 2 pneumoconiosis. Drs. Wheeler and Scott opined that the film was negative for pneumoconiosis, but Dr. Wheeler found it to be compatible with IP and Dr. Scott felt that it was most likely IP. All four physicians maintain excellent credentials for x-ray interpretation. I find the readings of Drs. Barrett and James more persuasive than Dr. Wheeler's and Dr. Scott's. All four doctors found some interstitial process present so that even the otherwise negative readings of Drs. Wheeler and Scott lend some credibility to positive readings of Drs. Barrett and James. Thus, I consider this x-ray positive.

Dr. Sharma read the September 3, 2001 x-ray as showing stable opacities in both lungs. It was not reread, and without more specificity, I do not consider this x-ray positive evidence of pneumoconiosis.

Dr. Branscomb read the January 18, 2002, August 1, 10, 11, 14, 15, 17, and 20, 2001, and the September 2, 3, and 5, 2001 x-rays. He found them to be either unreadable or negative. He thought the August 15, 2001 x-ray showed congestive heart failure. Most, if not all, of these films were taken when the miner was acutely ill in the hospital. Dr. Repsher also read the September 5, 2001 x-ray and found progressive development of severe interstitial fibrosis that he

later defined as UIP/IPF. Based on these readings, I do not find any of these films to be positive for pneumoconiosis.

I find the films that were not taken during the miner's hospitalizations the most persuasive ones. The films taken between January 1997 and August 2000 are the most probative. Of these 21 readings of 8 x-rays, 5 are by board-certified radiologists, 3 are by B-readers, and 8 are by dually certified readers. Of the board-certified radiologists who are not also B-readers, Dr. Baldwin read four x-rays and found densities on each of them. Dr. Sheya read an x-ray as positive. Of the B-readers who are not radiologists, Dr. Repsher found IP on one x-ray and considered the other unreadable, while Dr. James found an x-ray positive. Of the dually certified readers, Drs. Navani and Barrett found pneumoconiosis present, while Drs. Wheeler and Scott, who each read three x-rays, considered the films negative for pneumoconiosis but saw a progression of nodularity that they later described as IP.

I find the newly submitted evidence demonstrates a material worsening of the miner's condition pursuant to § 725.309, as interpreted by *Brandolino*, 90 F.3d 1502. All the prior x-rays were negative, while the majority of x-rays interpreted after the original claim was denied have shown an interstitial lung disease, and some of the physicians described it as pneumoconiosis.

Regarding a finding on the merits, I conclude that the most recent evidence of record is entitled to greater weight than the evidence that preceded the most recent denial. *Stanford v. Director, OWCP*, 7 BLR 1-541 (1984). Of the recent x-ray evidence, I place great weight on the readings of Drs. Navani and Barrett. I further find that the negative readings of Drs. Wheeler and Scott do not altogether negate the readings of Drs. Navani and Barrett because they described pulmonary fibrosis that, as described below, reflects a fibrosis caused by coal dust exposure. Accordingly, I also find that the x-ray evidence establishes the existence of pneumoconiosis pursuant to § 718.202(a)(1).

Evidence Under § 718.202(a)(4)

An adjudicator may find pneumoconiosis even with a negative x-ray when a physician has determined that the miner suffers from pneumoconiosis from clinical examination and laboratory test data, medical and work histories, and has given a reasoned basis for the medical opinion. 20 C.F.R. § 718.202(a)(4). Medical reports that are based upon and supported by patient histories, a review of symptoms, and a physical examination are adequately documented under the Regulations. *Justice v. Director, OWCP*, 6 BLR 1-1127 (1984). Where the physician's report fails to explain how the documentation supports the physician's conclusions, a judge may find the report is not "a reasoned medical opinion." *Smith v. Eastern Associated Coal Co.*, 6 BLR 1-1130 (1984). A medical opinion contradicted by objective medical data is insufficiently reasoned. *White v. Director, OWCP*, 6 BLR 1-368 (1983).

Medical Opinions

John K. Wright, M.D. examined the miner on June 7, 1979. DX 31. He recorded symptoms of a productive cough and shortness of breath; 30 years of coal mine employment that was still continuing and the miner's status as a non-smoker. On physical examination he found

the Claimant's lungs clear, with fair breath sounds. Based on a normal x-ray and a normal pulmonary function study he diagnosed no cardio-pulmonary disease .

Marlin Stahl, M.D. examined the miner on October 31, 1984. DX 32. He considered a work history of 38 3/4 years of underground coal mine employment; a family history significant for cancer; a medical history significant for pleurisy, attacks of wheezing, tuberculosis, chronic bronchitis, arthritis, myocardial infarction, angina, and knee surgery; a history of smoking less than one-half pack of cigarettes a day for 40 years before quitting in 1980. The miner described symptoms of a productive cough, wheezing, dyspnea, chest pain, orthopnea, and ankle edema. On physical examination of the lungs the doctor heard bilateral inspiratory rales.[2] He also considered the objective data from an x-ray, a pulmonary function study, and a blood gas study. He diagnosed industrial bronchitis and thought that the x-ray evidence suggested coal workers' pneumoconiosis. He added, however, that a better-penetrated film on deeper inspiration would be more helpful. He based this diagnosis on the x-ray, decreased pO2 values shown on laboratory testing, and normal FVC change with time. Dr. Stahl felt both diagnoses were related to coal dust exposure.

Dan Fennell, M.D. examined the miner on March 2, 1988. DX 33. He considered 39 3/4 years of coal mine employment, most recently as a belt man; an unremarkable family history; a medical history significant for knee surgery, a myocardial infarction, pneumonia as a child, wheezing with exercise, tuberculosis, and chronic bronchitis; a history of smoking less than one-half pack of cigarettes per day for 43 years before quitting in 1983; complaints of a productive cough, wheezing, shortness of breath, chest pain, orthopnea, and ankle edema; and a physical examination that revealed rales in the right mid-lung field. He also considered the results of an x-ray that showed enlargement of the heart (cardiomegaly), a normal pulmonary function study, and the mild hypoxemia shown on a blood gas study. Dr. Fennell diagnosed congestive heart failure based on a history of paroxysmal nocturnal dyspnea, orthopnea, hypoxemia, and limited ability to exercise; industrial bronchitis based on the persistent cough and sputum production despite a minimal smoking history; and coal workers' pneumoconiosis (CWP) based on his exposure history and x-ray consistent with CWP. Dr. Fennell attributed the congestive heart failure to coronary artery disease and the bronchitis/CWP to coal mine dust exposure with some contribution from having smoked cigarettes. He noted that the miner was retired and unable to work because of his angina. He added that the congestive heart failure was not adequately treated but felt that if it were his symptoms should improve. Dr. Fennell opined that the CWP contributed to a moderate extent to the miner's disability if the congestive heart failure were to resolve.

W. Snihurowych, M.D. saw the miner on May 15, 1990, May 21, 1990, and June 4, 1990, as evidenced by his office notes. EX 14. He examined him on those three occasions, noting a medical history significant for heart disease and knee surgery. Physical examination showed clear lungs and an enlarged prostate, and a biopsy of the prostate revealed benign hyperplasia.

Michael J. Lincoln, M.D., who is board certified in internal medicine and board eligible in pulmonary diseases, examined the Claimant on January 31, 1992. DX 27, 34. He considered 38 3/4 years of coal mine employment; an unremarkable family history; a medical history

[2] Wet, crackly lung noises heard on inspiration which indicate fluid in the air sacs of the lungs.

significant for pneumonia as a boy, arthritis, high blood pressure, knee surgery, and an acute heart attack; a history of smoking one pack of cigarettes a week for 38 years, ending in 1983; symptoms of a productive cough, wheezing, shortness of breath when walking around the house, chest pain, orthopnea, and occasional ankle edema; and a physical examination of the lungs that was normal. He read an x-ray as showing cardiomegaly, and a blood gas study and pulmonary function study each were normal. He diagnosed chronic bronchitis due to coal dust and smoking, based on a chronic cough and sputum production; and congestive heart failure due to acute myocardial infarction based on orthopnea and exertional dyspnea, in the absence of proof of lung obstruction by spirometry or blood gas study abnormalities. In his opinion, the miner was completely disabled by his angina and congestive heart failure, not by his chronic bronchitis.

Dr. Lincoln examined the miner again on September 17, 1994. DX 35. He considered 38 3/4 years of coal mine employment; a family history significant for high blood pressure; a medical history significant for pneumonia as a child, arthritis, a heart attack, high blood pressure, and knee surgery; a history of smoking a pack of cigarettes a week for 39 years, ending in 1984; symptoms of a productive cough, wheezing, shortness of breath, chest pain, orthopnea, and ankle edema; and on physical examination he heard no rales, rubs, rhonchi, or wheezing in the lungs. He also reviewed the results of an x-ray, a pulmonary function study, and a blood gas study. The last two yielded normal values. Dr. Lincoln diagnosed congestive heart failure based on his examination, history, and the x-ray that he attributed to coronary disease. He further diagnosed industrial bronchitis likely due to coal dust exposure. Dr. Lincoln opined that the Claimant was 100% impaired due to angina and congestive heart failure. He felt that none of the disability was due to the bronchitis he diagnosed.

Dr. Lincoln examined the Claimant for a third time on December 30, 1999. DX 26. He again considered an accurate coal mine employment history, all underground and mostly at the face of the mine; a family history significant for high blood pressure; the same medical history; a history of smoking one pack of cigarettes a week for 39 years before quitting in 1984; symptoms of daily shortness of breath, a cough with sputum and wheezing, chest pain, and two-pillow orthopnea, all of which had worsened since 1984; and on physical examination heard crackles in the lower one-third of the lungs bilaterally. Dr. Lincoln further considered the results of an x-ray, a pulmonary function study that was normal, and a blood gas study that was normal at rest but showed hypoxemia with exercise. He diagnosed pneumoconiosis by x-ray due to coal dust inhalation; borderline restriction due to obesity and/or pneumoconiosis; and exercise hypoxemia due to lung restriction. He opined that the miner had a mild to moderate impairment based on his symptoms and moderate-to-severe exercise hypoxemia. While he felt that arthritis has caused a very mild impairment, he had a problem apportioning the cause of disability between pneumoconiosis and coronary heart disease. Lacking any laboratory assessment of the miner's cardiac function, he made no apportionment.

On November 2, 1999, Max G. Morgan, M.D. examined the Claimant. DX 7. He had previously seen him on August 24, 1999, at which time he diagnosed cervical osteoarthritis, chronic obstructive pulmonary disease (COPD), pulmonary emphysema, pulmonary fibrosis, and probable pneumoconiosis, arteriosclerotic cardiovascular disease, and congestive heart failure, although this report is not part of the record. At the November evaluation, he considered a coal mine employment history; an unremarkable family history; a medical history significant for

arthritis, heart disease, knee surgery, peptic ulcer disease, and high cholesterol; a history of smoking four cigarettes a day for 20 years; complaints of a productive cough, wheezing, shortness of breath, chest pain, orthopnea, and ankle edema; and the results of a physical examination that revealed decreased breath sounds with no rales but a few wheezes. He also considered the results of an x-ray, a pulmonary function study that showed mild restrictive airway disease, and a blood gas study that demonstrated hypoxemia. He diagnosed a history of chest pain, arteriosclerotic cardiovascular disease, borderline cardiomegaly, congestive heart failure, and hypertension. He attributed these conditions to hyperlipidemia and hypertension. Dr. Morgan found a moderate impairment, adding that the miner was unable to walk up one flight of stairs before becoming dyspneic, he has chest pain with moderate exercise, and he has arthritis and disc disease. He attributed the impairment 30% to pulmonary disease, 40% to cardiac disease, and 30% to arthritis and disc disease.

Ben V. Branscomb, M.D., board certified in internal medicine and an expert in pulmonary diseases, provided a report dated March 8, 2000, based on a review of medical evidence. EX 3; 10. He considered 21 x-ray reports between February 1987 and December 1999, histories of smoking and coal mine employment, a medical history, subjective symptoms, seven pulmonary function and arterial blood gas studies between February 1988 and December 1999, and the reports of Drs. Lincoln, Fennell, and Morgan. He found no objective medical evidence of any pulmonary disease from any cause. The pulmonary symptoms the miner described—intolerance for exercise, chest pain, shortness of breath, cough, expectoration, orthopnea, and edema—were, in Dr. Branscomb's opinion, more descriptive of symptoms caused by well-documented heart disease. He did not find coal workers' pneumoconiosis or any lung disease associated with coal mine employment. He felt that the miner's total disability was due to severe cardiac disease, obesity, and orthopedic problems. Dr. Branscomb concluded that if the miner did not have heart disease, his pulmonary function would be fully normal. He also found no connection between the disability and coal dust exposure.

Dr. Branscomb reviewed medical evidence and provided another report about five months later, on August 18, 2000. EX 9. He considered 38 3/4 years of coal mine employment, the varying smoking histories, the miner's medical history, the many x-ray reports from August 1973 through December 1999, seven pulmonary function and blood gas studies administered between June 1979 and December 1999, and the reports of Drs. Wright, Stahl, Lincoln, and Morgan, as well as his own report of March 8, 2000. Dr. Branscomb found no evidence of CWP or pulmonary impairment. He diagnosed arteriosclerotic heart disease that had lead to two myocardial infractions, and which was complicated by angina pectoris, chronic biventricular congestive heart failure, and hypertension; hyperlipidemia; peptic ulcer disease; secondary erythrocytosis; possible prostatic cancer; and morbid obesity. His belief that the miner did not have pneumoconiosis was based on the lack of x-ray evidence, normal pulmonary function tests, and the fact that his hypoxemia was overwhelmingly likely to be the result of ventricular congestive heart failure. Dr. Branscomb opined that the miner was totally disabled as a result of his cardiac condition but not from pneumoconiosis. He attributed the disabling ischemic heart disease solely to genetics and the miner's life style factors.

When Dr. Branscomb reviewed Dr. James's report on October 11, 2000 he disagreed with several of Dr. James conclusions and did not alter his opinions. He stated that the U-shaped

opacities seen on x-ray are typically seen with both severe obesity and left ventricular failure, and are the least likely changes in CWP, which normally begins higher in the chest. He added that most of the best readers found the films negative for pneumoconiosis and that there was no consistency among those who read them as positive for the disease. Regarding the reduced pulmonary function values found on 9/4/00 compared to the 12/21/99 test, Dr. Branscomb felt that the fluctuation in the severity of left ventricular failure could explain the isolated reduction. While he agreed that CWP can be characterized by coarse opacities, it is classically and almost invariably characterized by predominantly small regular opacities in the upper middle or lower part of the upper lung zones. It is uncommon, he added, to have CWP in an early stage start at the base of the lungs and with solely irregular opacities. Dr. Branscomb stated that it would be particularly unusual for CWP to begin as rather coarse large opacities (category u). He stated that the miner's obesity, left ventricular heart disease with cardiomegaly, and altitude, not CWP, caused the hypoxemia. Finally, Dr. Branscomb believed that the academic medical papers Dr. James relied upon had little scientific merit and did not represent the generally accepted conclusions of experts in pneumoconiosis.

Dr. Branscomb reviewed additional medical evidence and provided a report dated November 13, 2003. EX 1. He considered a 9/19/84 pulmonary function test, Dr. Stahl's examination of 10/31/84, Dr. Fennell's examination, a 12/16/91 pulmonary function test, Dr. Lincoln's 1/31/92 report, Dr. Morgan's records from August and September 1999, Dr. Repsher's report of 12/5/00, the September 2001 hospital records, and readings of x-rays from 2/12/87, 12/16/91, 9/2/99, and 8/18/00. None of this new evidence altered his prior opinions. He thought that death was almost certainly the result of uncontrolled left ventricular failure with pulmonary edema, hypoxemia, and the progressive changes. He believed that it "would be absolutely impossible for a fatal obstructive lung disease to occur and result in death in the year 2001 in a person whose pulmonary function was entirely normal on 12/21/99." He found no reasonable basis for diagnosing chronic obstructive pulmonary disease. Dr. Branscomb believed that the miner's symptoms of streaky changes in the lower lungs, orthopnea, paroxysmal nocturnal dyspnea, progressive uncontrolled angina, and three myocardial infarctions were all caused by chronic left ventricular failure. He concluded that death was due to cardiac, not pulmonary disease.

Dr. Branscomb was deposed on October 18, 2005. EX 20. Regarding his review of the x-rays, he noticed that the markings changed—came and went—the way changes of heart failure do. Furthermore, he did not see any small, regular or irregular opacities. Dr. Branscomb explained that because all pulmonary function tests prior to 1999 were normal, the miner had no pulmonary impairment, and because he already had the symptoms of shortness of breath with exercise and angina, followed by three heart attacks, his symptoms were due to heart disease and obesity not to lung disease. He thought the loss of pulmonary function shown by Dr. James's and Dr. Repsher's pulmonary function studies demonstrated a restrictive process that would point either to congestive heart failure, pneumoconiosis, or pulmonary fibrosis. Because the miner last worked in 1984 and had excellent pulmonary function tests through 1999, then abruptly deteriorated and died within two years, the most likely explanation for the deterioration was a worsening of long-standing heart failure, or a new process that developed after 1999. He did not believe that any of the changes were due to coal-mine dust-induced lung disease based on the timing of the deterioration and the inconsistent x-ray evidence. Dr. Branscomb did not see a

basis for diagnosing usual interstitial pneumonitis (IUP) because he thought the findings were so characteristic of heart disease that it made no medical sense to speculate that another process caused the death. He acknowledged, however, that the miner could have developed fibrosis after 1999. He conceded that fibrosis can occur with coal dust exposure. In this case, however, he believed it could not be caused by coal dust because discrete opacities of the p, q, and r varieties should have shown up before the t and u opacities did, and that was never the case. Even assuming the existence of pneumoconiosis, Dr. Branscomb would still conclude that it had no effect on the miner's pulmonary function and caused no impairment because "the time intervals are wrong," meaning that it could not have remained in abeyance from 1984 to 1999 without causing any change in pulmonary function.

David S. James, M.D. examined the miner on August 18, 2000. CX 2. He is board certified in internal medicine, pulmonary disease, and critical care medicine. CX 1. He reviewed DX 1-36 and considered 40 years of coal mine employment including a detailed account of the exertional requirements of the miner's jobs; a medical history significant for coronary artery disease, two heart attacks, degenerative arthritis, and a crushed right knee that required surgery; symptoms of shortness of breath with cough and phlegm production, chest pain, wheezing primarily at night, and ankle edema. On physical examination he heard crackles on the right side and left side with wheezing or possible squawks on the left in different locations. He also reviewed the results of an x-ray, a pulse oxymetry to assess oxygen level at rest and after activity, and a pulmonary function study. Dr. James diagnosed: (1) coal workers' pneumoconiosis based on history of extensive dust exposure and an abnormal chest x-ray; (2) chronic bronchitis; (3) hypoxemia; (4) coronary artery disease; (5) hearing loss; and (6) osteoarthritis. He could think of no other diagnostic possibilities, other than coal workers' pneumoconiosis, to explain the current findings. He noted that irregular opacities can be seen in individuals with asbestosis, but the miner had no known asbestos exposure. He also thought that the x-ray findings were not consistent with congestive heart failure, and there was no clinical evidence of it according to Dr. Morgan's August 1999 examination. Dr. James believed that the prior x-rays showed findings similar to the current film, and it would be less likely that congestive heart failure would show such a consistent pattern. He also rejected idiopathic pulmonary fibrosis (IPF) as the condition shown by x-ray because it usually appears as small irregular opacities (s or t shaped) initially found in the lung bases. Dr. James relied upon a British study and a French study, found at CX 3 and 4, for the proposition that coal workers' pneumoconiosis can show up even years after exposure to coal mine dust ceases. Dr. James further opined that the hypoxemia was secondary to coal workers' pneumoconiosis because he found no other diagnoses likely to explain the condition. He felt that neither stable coronary artery disease nor stable congestive heart failure would cause arterial hypoxemia with exertion. He concluded that the miner was totally disabled and that coal workers' pneumoconiosis was a significant contributing cause of that disability, citing category 2 pneumoconiosis with a moderately severe decline in vital capacity and exercise-induced hypoxemia. He agreed that the coronary disease contributed to the miner's demise. Finally, Dr. James opined that the miner's condition worsened since 1999 as evidenced by the pulmonary function studies, and he attributed this worsening to CWP because of the absence of any other diagnosis likely to cause an abnormal x-ray, abnormal lung function tests, and abnormal blood oxygenation with exertion. He again stressed that the miner's cardiac status had been stable and there was no evidence of congestive heart failure on recent examinations.

Dr. James testified at the October 25, 2000 hearing. EX 8, p. 41-101. Prior to the hearing he had reviewed EX 1-5, the reports of Drs. Repsher and Branscomb. In his experience, about 5% of miners with pneumoconiosis have irregular opacities like this Claimant. In reviewing the x-ray reports of record, Dr. James noted that Dr. Lee's interpretation of the 10/4/94 film included irregular opacities in the lower lung zones. Dr. Wheeler also found irregular markings in the lower lung zones on the 12/21/99 x-ray. He also felt that Dr. Scott's findings on the 12/21/99 x-ray of moderate, non-specific linear interstitial fibrosis were consistent with his own interpretation of the film. He opined that the worsening of the miner's parenchymal process over an eight-year period was consistent with pneumoconiosis. Dr. James disagreed with Dr. Branscomb's statement that a worsening of x-ray results over time only occurs with complicated pneumoconiosis, and he cited British studies as support for his conclusion.

Dr. James testified that the miner's congestive heart failure had improved over the years, and that while he had hypoxemia due to the abnormalities shown on x-ray, it was consistent with pneumoconiosis. He further opined that the miner was disabled in 1999 and the pneumoconiosis was a significant factor in causing that disability. He disagreed with Dr. Branscomb's statement that the miner's pulmonary function was entirely normal. He pointed out that Dr. Branscomb did not have the most recent lung function studies to review and that a review of the tests over the last eight years of the miner's life showed a significant decline over time that was abnormal. He did not attribute the abnormality to obesity because the miner's weight had been relatively stable. He determined that pneumoconiosis was the cause of the decline in pulmonary function test values.

Dr. James also testified that there was no radiographic evidence of coal workers' pneumoconiosis in 1984. He agreed with Dr. Stahl's 1984 opinion of industrial bronchitis, which Dr. James equated to coal workers' pneumoconiosis. He also agreed with Dr. Fennell's diagnosis of bronchitis/coal workers' pneumoconiosis with contribution from smoking. He acknowledged, however, that the symptoms in earlier reports were the same as when he examined the miner.

Dr. James reviewed Dr. Repsher's report of November 29, 2000, and provided a supplemental report dated January 9, 2001 based on that review. CX 8. He disagreed with Dr. Repsher's diagnosis of chronic biventricular congestive heart failure, noting that there were no cardiac signs on either Dr. Repsher's or his own evaluations that were consistent with congestive heart failure such as jugular venous distention or abnormal additional heart sounds. He identified Dr. Repsher's findings of pitting edema and abnormal lung sounds as less specific and attributable to other conditions. Dr. James attributed them to the miner's diffuse lung disease, CWP. In addition, the November 2000 examination and EKG showed mild right ventricular enlargement. He found the evidence consistent with right-sided dysfunction secondary to diffuse lung disease due to coal dust exposure. When combined with lung disease, he stated, the combination was a form of cor pulmonale. Dr. James disagreed with Dr. Repsher's opinion that CWP does not progress after dust exposure ceases, and cited an article published in a peer-reviewed journal in support of that position. Dr. James's altered none of his opinions due to Dr. Repsher's report.

Dr. James provided a second supplemental report of October 16, 2005. CX 12. He reviewed Dr. Branscomb's November 2003 report and x-ray readings, Dr. Repsher's reports of 2000, 2003, and 2005, as well as his deposition, Dr. Barrett's x-ray report, the miner's final hospital records, and the death certificate. He again concluded that the miner's coal dust exposure was a significant contributing factor in the development of his fibrotic lung disease. He referred to a medical text, A. Churg and F.H.Y. Green, Pathology of Occupational Lung Disease, (Baltimore, Williams and Wilkins) at 129-207, that says chronic inhalation of coal dust is associated with irregular opacities and pulmonary fibrosis in some miners. Based on this, as well as the lengthy exposure to coal mine dust, and the clinical and radiographic evidence of his fibrotic lung disease, Dr. James thought it improper to characterize the pulmonary fibrosis as idiopathic. Once again, Dr. James disagreed that the miner had chronic biventricular congestive heart failure based on the echocardiogram of 11/29/00, which revealed normal left ventricular function, mild diastolic dysfunction, and pulmonary hypertension and right ventricular enlargement. He believed the last two conditions were "more likely than not" due to the miner's lung disease.

Lawrence Repsher, M.D. , who is board certified in internal medicine, pulmonary disease, and critical care medicine, examined the miner on November 29, 2000 and provided a report dated December 5, 2000. EX 6; EX 7. He considered symptoms of a chronic productive cough, progressive dyspnea on exertion that has worsened over the last two years, orthopnea, and ankle edema; a history of smoking less than one pack of cigarettes per week for 34 years before quitting at the age of 50; and a physical examination that revealed bilateral rales in the lungs. He also reviewed the results of an ECG, a blood gas study, and a pulmonary function study. He diagnosed coronary artery disease complicated by angina pectoris and chronic biventricular congestive heart failure; hypertension of unknown cause complicated by hypertensive cardiovascular disease; secondary erythrocytosis; and marked abdominal obesity. He found no evidence of coal workers' pneumoconiosis or intrinsic lung disease.

Dr. Repsher criticized Dr. James's report on several bases. Dr. Repsher asserted that the article quoted by Dr. James, by Vallyathan, et al., in no way indicates or suggests that irregular opacities can occur as a result of coal dust exposure. He stated that irregular opacities occur in 4-5% of non-smoking non-miners and in the majority of smokers. He stated that s and t opacities do not suggest that coal mine dust is the cause of such opacities. Furthermore, he added that u-shaped opacities are due to congestive heart failure. In Dr. Repsher's opinion, the miner's productive cough was due to left ventricular congestive heart failure. He felt that industrial bronchitis generally resolves 6-12 months after exposure ends. Dr. Repsher was confident that the miner's hypoxemia, two-to-three-pillow orthopnea, and almost nightly paroxysmal nocturnal dyspnea were due to his congestive heart failure. Whereas Dr. James found no other diagnostic possibilities for his findings, Dr. Repsher responded that all the miner's symptoms and the findings based on the x-rays, PFTs, and ABGs were explained by the miner's cardiomyopathy that resulted in chronic left ventricular congestive heart failure. Dr. Repsher further criticized Dr. James's reliance on a British study and a French study. He also asserted that the miner's cardiopulmonary condition worsened with time and that the cause was a worsening of his underlying coronary and hypertensive cardiovascular diseases. Dr. Repsher opined that the miner was totally disabled from coal mine work but he found no evidence of CWP, and thus ruled it out as a cause.

Dr. Repsher reviewed medical records that Dr. Branscomb had in his November 13, 2003 report, as well as the death certificate. EX 2. In a written opinion dated November 20, 2003 Dr. Repsher commented that the miner's medical records never documented pulmonary fibrosis. He thought the x-ray changes were due to chronic congestive heart failure with intermittent exacerbations. The only other basis he could see for a diagnosis of CWP was the coal mine employment history. He found no evidence of coal workers' pneumoconiosis or any other pulmonary or respiratory disease caused or aggravated by coal mine employment. Dr. Repsher gave four reasons for his conclusion: (1) the absence of x-rays finding of CWP; (2) persistently normal pulmonary function tests; (3) the absence of blood gas evidence of CWP; and (4) the cause of death was chronic left ventricular congestive heart failure that flooded the lungs with fluid (pulmonary edema), eventually causing death from respiratory failure.

Dr. Repsher wrote another report dated February 1, 2005, based on a review of x-rays taken from 1/21/97 through 9/5/01. EX 5. The x-rays from 1/21/97 through 8/17/01 proved to him a progressively developing and ultimately severe case of usual interstitial pneumonitis/idiopathic pulmonary fibrosis (IP/IPF), a uniformly fatal disease that is relatively uncommon in the general population. He thought the ground glass opacities and severe honeycombing he saw on the 8/15/01 CT scan were classic signs of UIP/IPF. Its cause is unknown, but has "never been related to work as a coal miner or exposure to coal mine dust." He believed the miner was totally disabled, but without x-ray evidence of CWP he would not attribute the disability to coal mine employment.

Dr. Repsher explained at his September 12, 2005 deposition (EX 19) that a diagnosis of pulmonary fibrosis is more general than usual interstitial pneumonitis and includes other conditions. In his opinion, the miner's appearance and clinical course clearly pointed to the diagnosis of UIP/IPF. The condition is not found in any particular occupation, although it occurs somewhat more in men than women. Upon examination of the miner, Dr. Repsher found a serious restrictive pulmonary defect that he attributed to more severe UIP/IPF. It had worsened considerably in just three months, and, according to Dr. Repsher, coal mine dust-induced lung disease does not progress that quickly. He attributed the miner's symptoms of coughing, paroxysmal nocturnal dyspnea, orthopnea, ankle edema, and severe shortness of breath to serious left ventricular heart failure. Regarding the literature cited by Dr. James, Dr. Repsher pointed out that there had been no control for whether the study participants smoked cigarettes, which rendered the conclusions unscientific. Dr. Repsher reiterated his opinions regarding disability causation, adding that even if the miner were found to have simple CWP, it did not cause his disability because it does not cause significant impairment in lung function "on the average." Both congestive heart failure and UIP/IPF explain the disability.

On cross-examination, Dr. Repsher explained that in looking back at the evidence, he would now say that in December 2000, the miner's respiratory impairment was possibly due to his fatal, intrinsic lung disease of UIP/IPF, and not just his heart disease. He would also change his opinion from November 2003 that there was no evidence of pulmonary fibrosis. He now feels it may have been due to early UIP/IPF. Early on, he explained, changes from congestive heart failure look the same as those from UIP/IPF. Dr. Repsher opined that the miner was crippled by pulmonary fibrosis for the last couple of years of his life and died directly as a result of it. Later, Dr. Repsher testified that the miner could have died of his severe heart disease

before he was dying of his UIP/IPF, or they both contributed to his death. Dr. Repsher also altered his prior opinion that respiratory failure was in no way related to any lung disease but was secondary to his chronic left ventricular congestive heart failure. He explained that at the time he made that assessment he was unaware of the UIP/IPF. Once he read a CT scan and x-rays that showed the UIP/IPF, he changed his opinion.

Death Certificate

When the miner died on September 9, 2001 Daniel C. Monahan, M.D., signed the death certificate, listing the cause of death as pulmonary fibrosis. DX 39, 42, CX 11.

Hospital Records

There are 211 pages of hospital records from September 1, 2001 to the date of the miner's death on September 9, 2001. DX 45. They show that he was attended by Dr. David A. Nichols and his family physician, Dr. Monahan, who examined him on September 2, 2001. He presented with a diagnosis of pulmonary fibrosis. Dr. Nichols noted a significant decline in pulmonary status over the previous two months. The miner was on oxygen, bronchodilators, antibiotics, steroids, and morphine but eventually died on September 9, 2001. The discharge diagnoses were: (1) pulmonary fibrosis; (2) CWP; (3) chronic obstructive pulmonary disease; (4) hyperlipidemia; (5) cor pulmonale; and (6) coronary artery disease.

Prior to the most recent denial, the record contained the medical opinions of Drs. Wright, Stahl, Fennell, Snihurowych, and Lincoln. Dr. Snihurowych examined the Claimant for urological concerns, so I place no weight on his opinion. Dr. Wright found no cardio-pulmonary disease. Dr. Stahl diagnosed industrial bronchitis and believed the x-ray suggested CWP. I consider this equivalent to a finding of CWP. Dr. Fennell diagnosed CWP, and Dr. Lincoln diagnosed chronic bronchitis due in part to coal dust exposure. This is also equivalent to a finding of CWP. I find all four opinions to be well documented because they contain physical examinations, accurate smoking histories, and a report of coal mine employment. *Perry v. Director, OWCP*, 9 BLR 1-1 (1986). Dr. Wright's opinion is supported by the x-ray on which he relied. It was read by one board-certified radiologist and one dually certified reader as negative. However, his evaluation occurred five years prior to the next examination (Dr. Stahl's) and fifteen years before Dr. Lincoln's examination. I place less weight on Dr. Wright's opinion and more weight on the more recent evaluations because they contain additional information and so are more likely to accurately assess the miner's condition. *Gillespie v. Badger Coal Co*., 7 BLR 1-839 (1985).

Dr. Stahl's opinion is inconsistent with the x-ray on which he relied. Both Dr. Bauermeister and Dr. Sargent found the film negative, and these doctors possess expert credentials in radiographic interpretation. Dr. Stahl, however, felt the x-ray suggested CWP but suggested that a better-penetrated film would be helpful. He also relied upon a "normal FVC change with time" to make his diagnosis, but he did not explain what he meant by this, and I find it unclear. He further relied upon a decreased pO2 as pointing to CWP, but the blood gas study evidence was normal until many years later. Accordingly,

although there is adequate documentation, Dr. Stahl's opinion is not well reasoned based upon the underlying objective evidence, so I discount it.

Dr. Fennell's diagnosis of CWP was based upon the miner's exposure history and x-ray. However, the x-ray read by Dr. Sargent that preceded Dr. Fennell's examination was found negative, and there is no record of any other x-ray taken around the time of Dr. Fennell's evaluation. Consequently, I find no basis for Dr. Fennell's conclusion that the x-ray evidence supports a finding of CWP. Furthermore, a diagnosis of CWP based only on an exposure history and x-ray can be accorded less weight because it amounts to no more than an x-ray reading. *Cornett v. Benham Coal, Inc.*, 227 F.3d 569 (6th Cir. 2000). Dr. Fennell based his finding of industrial bronchitis, which is legal pneumoconiosis, on the miner's persistent cough and sputum production despite a minimal smoking history. I find this diagnosis to be better supported because the miner had stopped smoking four to five years before this examination yet continued to have a productive cough. Therefore, I place some weight on Dr. Fennell's diagnosis of legal pneumoconiosis.

Dr. Lincoln had the opportunity to examine the miner in both 1992 and 1994. On both occasions he diagnosed chronic bronchitis due, at least in part, to coal dust exposure. However, the x-rays of record taken closest in time to Dr. Lincoln's examinations were read as negative or unremarkable. While he based his diagnosis on the miner's chronic cough and sputum production, the objective evidence did not support his conclusion, and he accounted for the other symptoms by diagnosing congestive heart failure. In his 1994 report, Dr. Lincoln did not explain why he diagnosed industrial bronchitis when the x-ray was read as showing congestive heart failure but not pneumoconiosis, and he attributed the miner's symptomatology to his heart disease. Consequently, I discount Dr. Lincoln's opinion.

Based on these four medical opinions, the only opinion I find entitled to any weight was Dr. Fennell's. Although the evidence is weak, I would have found, by a slight preponderance of the evidence, that the miner had established the existence of pneumoconiosis pursuant to § 718.202(a)(4). This hardly means that later evidence could not establish a material worsening of his condition.

Five physicians provided opinions after the claim first was denied. Dr. Lincoln and Dr. James diagnosed pneumoconiosis. Dr. Morgan did not address the existence or absence of pneumoconiosis. Dr. Branscomb and Dr. Repsher opined that the miner did not suffer from pneumoconiosis, although Dr. Repsher found UIP/IPF. During the miner's final hospitalization, Drs. Nichols and Monahan diagnosed pneumoconiosis.

Dr. Lincoln's report is well documented but the two December 1999 x-rays were found negative for pneumoconiosis by the three physicians whose readings are in the record. Drs. Wheeler and Scott found IP and Dr. Branscomb diagnosed congestive heart failure based on the radiographs. Because Dr. Lincoln's finding of pneumoconiosis was based on an x-ray, and the readings of record do not support that finding, I do not consider his opinion to be well reasoned. *Fuller v. Gibraltar Corp.*, 6 BLR 1-1291 (1984). Accordingly, I discount it.

Dr. Morgan did not diagnose pneumoconiosis, but, more specifically, he did not address the presence or absence of the disease. Therefore, I place no weight on his opinion on this issue. I note, however, that he relied on a smoking history that was much less extensive than all the other reported smoking histories of record, and an erroneous smoking history can make an opinion a poorly documented one. *Stark v. Director, OWCP*, 9 BLR 1-36 (1986). Therefore, even if I considered his finding of "probable pneumoconiosis" from the August 24, 1999 evaluation that does not appear of record, as equivalent to a diagnosis of CWP, I would discount it because of the erroneously low smoking history.

Dr. Branscomb is an undisputed expert in the field of pulmonary medicine. *Wetzel v. Director, OWCP*, 8 BLR 1-139 (1985). He also reviewed all the medical evidence of record, thus providing him with a broad base of information on which to reach his opinion. I find that his unwillingness to consider a cause other than heart disease for the miner's symptoms, despite a clear advancement of markings of pulmonary disease on the x-rays over time, weakens the persuasiveness of his opinion. While he found that the markings came and went, thus pointing to heart failure, Dr. James interpreted them consistent over time, therefore pointing away from cardiac disease. In fact, a review of the x-ray reports beginning in 1994 supports Dr. James's conclusion. Although Dr. Branscomb consistently read the x-rays as negative, every doctor but Dr. Sheya in 1994, detected some pulmonary process. Dr. Branscomb also admitted that one of the potential causes for it was an ongoing restrictive lung process, as demonstrated by the 2000 pulmonary function studies. He also acknowledged that the miner could have developed pulmonary fibrosis after 1999, and he testified that fibrosis can be associated with coal dust exposure. His unwillingness to link the fibrosis to coal dust inhalation was based upon his belief that discrete p, q, or r opacities should have been visible by x-ray prior to the u-shaped opacities that appeared after 1999. Dr. James, however, cited medical literature that supports the proposition that CWP is progressive and can appear years after the cessation of coal mine employment—the position Congress and the Secretary have taken. I find that because Dr. Branscomb did not foreclose all possibility that the miner's restrictive pulmonary fibrosis may have been due to coal dust inhalation, and because of his professed reluctance to consider a cause other than heart failure for the miner's symptoms, his opinion is less persuasive than that of Dr. James.

Drs. Repsher and James possess excellent qualifications in the fields of internal and pulmonary medicine. *Scott v. Mason Coal Co.*, 14 BLR 1-38 (1990). I do not credit one opinion over the other based on credentials. Their opinions are based not only on their examinations of the miner, but also a review of essentially all the medical evidence in this voluminous record. Thus, they both had a large data set to draw upon. One reason I discount Dr. Repsher's opinion is because he did not consider an x-ray in conjunction with his November 2000 evaluation of the miner, and Drs. Barrett and James both found the August 18, 2000 x-ray positive for pneumoconiosis. This could have influenced his opinion, although frankly I doubt it would have made any difference to him. More troubling is Dr. Repsher's unshakable confidence, in his first review of the medical evidence, that all of the miner's symptoms were explained by congestive heart failure.

Once he reviewed the most recent pulmonary function studies, noting the miner's precipitous decline, and compared all the x-ray reports, he changed his mind and was then certain that UIP/IPF accounted for the findings of x-ray and CT scan, as well as the restrictive pulmonary defect, and some of the miner's symptoms. He ruled out CWP because he thought coal mine-dust-induced lung disease does not progress as fast as the condition that the miner had. Based on his change of opinion, despite his expressed certainty in each case, I find that his credibility is compromised.[3] Moreover, Dr. Repsher's opinion that CWP does not progress as fast as the miner's lung condition did is aptly contradicted by Dr. James, whose opinion I find more persuasive. For these reasons, I place less weight on Dr. Repsher's opinion.

Dr. James's opinion is supported by the x-ray reading of Dr. Barrett, a dually certified reader. The interpretations of UIP/IPF also support Dr. James's opinion in that the reviewing radiologists were not necessarily aware of the miner's exposure history. What I find most persuasive about Dr. James's opinion is that he took into account the miner's extensive and lengthy exposure to coal mine dust. I accept his conclusion that because of this exposure, it does not make sense to describe the pulmonary fibrosis as idiopathic; a very clear possible, and even probable, cause for it appears in the record: the extensive coal mine employment history. All the miner's 40 years of coal mine work were underground, and most were at the dustiest part of the mine. Also supporting Dr. James's opinion are the consistent findings of irregular lung markings beginning with the 1994 x-rays. Furthermore, his belief that CWP can manifest years after coal mine dust exposure ends is supported by the medical literature he cited, as well as his own experience, that about 5% of miners with pneumoconiosis have irregular opacities like those seen on these x-rays. The new regulations recognize that pneumoconiosis is a latent and progressive disease that may first become detectable only after the cessation of coal mine dust exposure. 20 C.F.R. § 718.201(c) (2005). Dr. James ruled out worsening congestive heart failure, enumerating symptoms of the disease that were not present when either he or Dr. Repsher examined the miner in 2000. He also pointed to the miner's stable weight, albeit heavy, and stable heart condition, as further evidence that the worsening in the miner's condition was pulmonary in nature. For these reasons, I find Dr. James's opinion to be the most persuasive and therefore place the greatest weight on it.

The September 2001 hospital records show the diagnoses of both CWP and pulmonary fibrosis. Dr. Monahan was the miner's treating physician who attended the miner during his final week of life. I place some weight on the hospital records, and Dr. Nichols's and Dr. Monahan's diagnoses, because they examined the miner, attended him every day from September 2-9, 2001, and considered numerous x-rays, a medical history, and the symptoms of the miner's final illness. Because their reports are part of the hospital records, I also find that they are completely unbiased. They tend to buttress Dr. James's opinion.

[3] This conclusion is based on the record in this case, not on a general conviction that experts who reconsider their opinions are wafflers unworthy of belief. To the contrary, the ability to reconsider firmly held views in light of additional data often enhances an expert's persuasiveness.

For the foregoing reasons, I place the greatest weight on Dr. James's opinion, as supported by the hospital records, and conclude that the medical opinion evidence establishes the existence of pneumoconiosis. I also conclude that the evidence demonstrates a material worsening in the miner's condition since the prior denial.

Total Disability Causation

Claimant must establish by a preponderance of the evidence that his total disability is due to pneumoconiosis. *Baumgartner v. Director, OWCP*, 9 BLR 1-65, 1-66 (1986); *Gee v. Moore & Sons*, 9 BLR 1-4, 1-6 (1986) (*en banc*). The Board requires that pneumoconiosis be a "contributing cause" to the miner's disability. *Scott v. Mason Coal Co.*, 14 BLR 1-37 (1990) (en banc), *overruling Wilburn v. Director, OWCP*, 11 BLR 1-135 (1988). Because this claim was filed prior to the enactment of the new regulations, the Tenth Circuit law prior to that time controls. The Tenth Circuit requires that the pneumoconiosis be "at least a contributing cause." *Magnus v. Director, OWCP*, 882 F.2d 1527, 1531 (10th Cir. 1989).

Prior to the most recent denial, Drs. Wright and Snihurowych did not address the causation issue. Dr. Stahl did not assess any disability or its cause. Dr. Fennell thought that the miner was unable to work because of angina, but he believed that pneumoconiosis contributed to a moderate extent to that disability. Dr. Lincoln asserted that the miner was totally disabled by his angina and congestive hart failure but not by his chronic bronchitis. Accordingly, the only opinions to consider are those of Drs. Fennell and Lincoln.

Both physicians' opinions are well documented, as both examined the miner, took medical, occupational, and smoking histories, and considered the results of PFTs and ABGs. I place less weight on Dr. Fennell's opinion because his examination of the miner took place so long ago, four years before Dr. Lincoln first examined the miner and six years before Dr. Lincoln's second evaluation. The normal results on PFT and ABG for the last two exams negated one of the reasons for Dr. Fennell's opinion: mild hypoxemia based on the 1988 blood gas study. Furthermore, because I have discounted Dr. Fennell's diagnosis of CWP based on the x-ray readings that did not support his conclusion, I do not credit his conclusions about what may have caused that flawed diagnosis.

Dr. Lincoln's opinion is well documented and reasoned. Based on the objective studies he considered, the miner's medical history that included a recent myocardial infarction, and the presenting symptoms, I place great weight on his opinion. Consequently, I find that the evidence prior to the most recent denial did not establish that the miner's pneumoconiosis, if he had had it, was at least a contributing cause of his total disability.

The hospital records and death certificate do not address disability causation. Dr. Lincoln opined that the miner had a mild to moderate impairment but he thought there was insufficient data to apportion the cause between pneumoconiosis and coronary heart disease. Dr. Morgan found a moderate impairment and apportioned 30% of it to pulmonary disease. Dr. Branscomb felt the miner was totally disabled but believed the cause to be severe cardiac disease, obesity, and orthopedic problems. He found no connection between disability and coal dust exposure, even assuming the existence of pneumoconiosis. Dr. Repsher also found the miner to be totally

disabled but ruled out pneumoconiosis as a cause. He attributed the disability to congestive heart failure and UIP/IPF. Dr. James opined that the miner was totally disabled and that pneumoconiosis was a significant contributing cause of that disability, with coronary disease contributing as well.

Although I discounted Dr. Lincoln's opinion on the issue of pneumoconiosis, that diagnosis is consistent with my finding. Therefore, I place more weight on his opinion regarding the cause of the miner's total disability. This opinion is well reasoned given the existence of the disease, the development of hypoxemia since Dr. Lincoln's 1994 examination, and the changes on physical examination. Consequently, I place some weight on Dr. Lincoln's opinion and find that it supports that of Dr. James.

I discount Dr. Morgan's opinion because he did not specify what pulmonary disease the miner had. In fact, he made no diagnosis of any pulmonary disease (COPD, bronchitis, pneumonia, emphysema, or pneumoconiosis). I find no objective basis for his conclusion.

I also discount Dr. Branscomb's opinion. He steadfastly maintained that the miner had cardiac failure and refused to consider another process as the cause of the symptoms of exercise intolerance (i.e., of arterial hypoxemia with exertion) or of the test results. Dr. Branscomb did not diagnose CWP. A medical opinion that finds that a claimant does not have CWP "can carry little weight" in assessing the cause of the miner's total disability "unless the ALJ can and does identify specific and persuasive reasons for concluding that the doctor's judgment on the question of disability causation does not rest upon h[is] disagreement with the ALJ's finding as to either or both of the predicates . . . in the causal chain." *Toler v. Eastern Assoc. Coal Co.*, 43 F.3d 109 (4th Cir. 1995). When Dr. Branscomb assumed the existence of pneumoconiosis, he stated that his opinion about total disability causation would not change. He doubted the pneumoconiosis would have been present from 1984 to 1999 yet cause few symptoms, and he doubted that abrupt deterioration in pulmonary function would be due to CWP. The "tenet that pneumoconiosis is non-progressive is simply inconsistent with the 'assumption of [disease] progressivity that underlies much of the statutory regime.'" *Consolidation Coal Co. v. Director, OWCP [Kramer]*, 305 F.3d 203 (3rd Cir. 2002). An opinion that simple pneumoconiosis cannot be totally disabling is considered hostile to the Act. *Kaiser Steel Corp. v. Director, OWCP*, 748 F.2d 1426 (10th Cir. 1984). Because I cannot identify persuasive reasons for concluding that Dr. Branscomb's opinion does not rest upon his belief that the miner did not have CWP, I place less weight on his opinion. I also note that when Dr. Branscomb was deposed he acknowledged that fibrosis could have developed after 1999 and that it could be caused by coal dust exposure. Thus, indirectly, he did not foreclose the possibility that the worsening of the pulmonary function studies could have been due to CWP.

I find Dr. James's opinion highly persuasive. I find his reasoning logical and consistent with the objective medical evidence and Claimant's clear decline in the last two years of his life that coincided with worsening x-rays and pulmonary function studies. Dr. James's conclusion also takes into account, rather than ignores, the miner's extensive coal mine employment history. Accordingly, I place great weight on his opinion.

Dr. Repsher ultimately opined that the miner's total disability was due to his congestive heart failure and UIP/IPF. He believed the miner was crippled by his pulmonary fibrosis for the last two years of his life. Assuming the existence of simple pneumoconiosis, he opined that it did not cause any disability because "it does not cause significant impairment in lung function on the average." Because Dr. Repsher did not foreclose all possibility that simple pneumoconiosis can be totally disabling, I do not consider his opinion hostile to the Act. I have, however, found that the miner's pulmonary fibrosis was not idiopathic, as Dr. Repsher insisted, but is readily explained by years of inhaling coal mine dust. With Dr. Repsher's opinion as to the causation of the pulmonary fibrosis set aside, his opinion, in fact, supports the miner's claim, and I find it well reasoned. The medical records demonstrate a clear deterioration in pulmonary ability beginning in 1999, and the x-ray evidence supports the finding of worsening pulmonary fibrosis. Accordingly, I place great weight on this portion of Dr. Repsher's opinion.

Based on the foregoing analysis, I place the most weight on the opinion of Dr. James, as supported by those of Drs. Repsher and Lincoln. Therefore, I find that the newly submitted evidence establishes a material worsening in the miner's condition regarding the issue of total disability causation, pursuant to § 725.309. I further find the evidence establishes, by a preponderance of the evidence, that the miner's pneumoconiosis contributed in part to his total respiratory disability. Because I have found a material change in conditions with respect to §§ 718.202(a) and 718.204(c), I will now address the merits of entitlement.

The Existence of Pneumoconiosis

I find the more recent evidence to be more probative than that assembled in conjunction with the earlier decision to deny the claim. Based on the foregoing analysis of the x-ray and medical opinion evidence, I find that the evidence establishes the existence of pneumoconiosis under both § 718.202(a)(1) and (a)(4).

Arising Out of Coal Mine Employment

In order to be eligible for benefits under the Act, Claimant must prove that the miner's pneumoconiosis arose, at least in part, out of his coal mine employment. § 718.203(a). As Judge Teitler credited the miner with at least 40 years of coal mine employment, and this finding was affirmed as unchallenged on appeal, he is entitled to the rebuttable presumption set forth in § 718.203(b) that his clinical pneumoconiosis (*i.e.,* CWP) arose out of coal mine employment. *See, Anderson v. Director, OWCP,* __ F.3d __, 2006 WL 2053841 at *3 (10th Cir. 2006). I find that the presumption has not been rebutted because despite Dr. Navani's opinion that the parenchymal changes were more likely due to asbestos than coal dust, there is no evidence that the miner had any asbestos exposure. Furthermore, as stated above, I have found that the remaining physicians' diagnoses of UIP/IPF actually represented pulmonary fibrosis due to coal dust exposure because the condition was not ideopathic.

Total Disability

Employer did not appeal the finding of total disability to the Benefits Review Board. However, the Board held that Judge Teitler erred in his consideration of the newly submitted

pulmonary function and blood gas study evidence in that he failed to consider Dr. Repsher's comments questioning the reliability of the tests and in mechanically relying on the most recent pulmonary function study without explaining why he considered it more credible than the contemporaneous studies. Thus, the Board directed that I reconsider the objective evidence pertaining to total disability on remand.

Subsection 718.204(c)(1) provides that total disability may be established if the pulmonary function study evidence produces qualifying values. The record contains pulmonary function studies taken on 12/16/94 by Dr. Morgan; 9/2/99 by Dr. Morgan; 12/21/99 by Dr. Lincoln; 8/18/00 by Dr. James; and 11/29/00 by Dr. Repsher. DX 5, DX 6, DX 26, CX 5, EX 8. They are summarized in the Decision and Order – Awarding Benefits of November 16, 2001. CX 9. Only the last of these five studies yielded qualifying values. Dr. Repsher stated in his December 5, 2000 report that the test could not be interpreted due to the miner's poor inspiratory efforts. EX 6. Accordingly, he invalidated the only qualifying study. Therefore, I find that the PFT evidence does not establish, by a preponderance of the evidence, the existence of total disability under subsection 718.204(c)(1).

Total disability can be demonstrated under § 718.204(c)(2) if the results of arterial blood gas studies meet the requirements listed in the tables found at Appendix C to Part 718. There are three blood gas studies submitted in connection with this case. They are Dr. Morgan's study of 9/2/99, Dr. Lincoln's study of 12/21/99, and Dr. Repsher's study of 11/29/00. DX 10, DX 28, EX 6. The 9/2/99 study produced qualifying values after exercise. The December 21, 1999 study yielded qualifying values both at rest and after exercise, and the November 29, 2000 study also produced qualifying values. Dr. Kennedy validated the two 1999 studies. Based on a preponderance of the evidence, I find that the blood gas study evidence establishes total disability pursuant to § 718.204(c)(2).

Total disability can be established under § 718.204(c)(3) if there is evidence of cor pulmonale with right-sided congestive heart failure. Cor pulmonale was diagnosed Dr. Nichols as a discharge diagnosis of September 9, 2001. However, he did not also make the diagnosis of right-sided congestive heart failure. Dr. James also diagnosed cor pulmonale with right ventricular enlargement and right-sided dysfunction secondary to diffuse lung disease caused by coal dust exposure. Because Dr. James did not state that his findings of right ventricular enlargement or ride-sided dysfunction were the same as right-sided congestive heart failure, I cannot make that equivalency either. Accordingly, I find that the preponderance of the evidence does not establish the existence of cor pulmonale with right-sided congestive heart failure pursuant to § 718.204(c)(3).

Finally, total disability can be established under § 718.204(c)(4) if a physician exercising reasoned medical judgment, based on medically acceptable clinical and laboratory diagnostic techniques, concludes that a miner's respiratory or pulmonary condition prevents him from engaging in his usual coal mine work or comparable and gainful work.

Prior to the most recent denial, Dr. Wright, in 1979, found no cardio-pulmonary disease and did not express an opinion as to disability. Dr. Stahl, in 1984, did not address the issue of disability either. Dr. Fennell examined the miner in 1988 but did not specify to what extent he

thought the miner was disabled. Dr. Snihurowych did not address this issue. Dr. Lincoln found total disability in 1992 and 1994 but attributed it completely to angina and congestive heart failure. Based on the chronological remoteness of these examinations, they are not as likely to represent an accurate evaluation of the miner's condition in his later years. *Gillespie v. Badger Coal Co.*, 7 BLR 1-839 (1985); *see also Bates v. Director, OWCP*, 7 BLR 1-113 (1984) (more recent reports of record are entitled to more weight than reports eight years earlier). I find these reports less persuasive and accordingly place no weight on them.

Beginning in 1999, Dr. Lincoln found the miner to have a mild-to-moderate pulmonary impairment. Dr. Morgan also found a moderate impairment but added that the miner could not walk up one flight of stairs before becoming short of breath and that he had chest pain with moderate exercise. Based on the exertional requirements of the last coal mining job at the face of the mine, I consider this opinion equivalent to one of total disability because those limitations would prevent a person from performing that job. Dr. Branscomb declared the miner totally disabled after his examination in March 2000. Dr. James found the miner to be totally disabled from a pulmonary perspective. Dr. Repsher opined that the miner was totally disabled from a respiratory standpoint.

With the slight exception of Dr. Lincoln's opinion, the remaining physicians found the miner to be totally disabled. I find these opinions to be well documented and reasoned in light of the miner's unwavering symptomatology, his clinical presentation, and the worsening of his condition with time as evidenced by the PFTs, ABGs, and x-rays. Accordingly, I find that the medical opinion evidence establishes, by a preponderance of the evidence, that the miner was totally disabled pursuant to § 718.204(c)(4).

Total Disability Causation

In addressing this issue on the merits, I defer to the medical opinions beginning in 1999 because it was at that time that the miner's condition precipitously declined, and any earlier medical opinions would not accurately reflect that worsening of condition. I adopt my prior analysis of the evidence on this issue and place the greatest weight on Dr. James's opinion, as supported by Dr. Lincoln and Dr. Repsher. Therefore I find that the miner's coal workers' pneumoconiosis was at least a contributing cause of his total respiratory disability. Accordingly, the Claimant is entitled to benefits.

Conclusion

The Claimant has met her burden to establish the existence of total disability due to pneumoconiosis. Consequently, she is entitled to benefits under the Act.

Attorney Fees

No award of attorney's fees for services to the Claimant is made, for no application has been made for them. The Claimant's counsel is granted 30 days to submit an application that complies with §§ 725.365 and 725.366 of the Regulations and that is served on all parties,

including the Claimant. The employer has ten days following receipt of the application in which to file any objections.

ORDER

The claim of the survivor of the miner for black lung benefits under the Act is GRANTED, and it is ORDERED that the employer, Energy West Mining Company, now doing business as Utah Power & Light, Mine Division, shall pay to the Claimant all augmented benefits to which she is entitled under the Act, commencing June 1, 1999.

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WILLIAM DORSEY
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).